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Author(s): Susan Kent

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THE INFLUENCE OF SEDENTISM AND AGGREGATION ON POROTIC HYPEROSTOSIS AND ANAEMIA: A CASE STUDY

SUSAN KENT

*Old Dominion University
Norfolk, Virginia*

Porotic hyperostosis and iron deficiency anaemia are often thought to be the direct result of a low iron diet, and by implication, a diet low in meat. A case study using the prehistoric Anasazi from the North American Southwest, however, shows diet to be virtually inconsequential in the rise in frequency of porotic hyperostosis and chronic iron deficiency anaemia. Instead, it is shown that the effects of sedentism and aggregation result in an increase in viral, bacterial and parasitic diseases, all of which can significantly contribute to the incidence of porotic hyperostosis by causing chronic iron deficiency anaemia. The increasing frequency of this type of anaemia in the Southwest and elsewhere through time is a direct result of the rise of sedentary aggregated communities.

What do skeletal populations from the southwestern part of the United States have in common with those from Subsaharan Africa, Egypt, Greece, Çatal Hüyük in Turkey and the Yucatan Peninsula of Mexico? Each population contains skulls that exhibit varying degrees of porotic hyperostosis. Porotic hyperostosis is a descriptive term for the cranial lesions that are characterised by a sieve-like porosity involving parts of the outer skull (Moseley 1965; 1966: 121). The explanation often given for the presence of porotic hyperostosis on skulls from Greece and the early Neolithic village of Çatal Hüyük, Turkey is thalassaemia, one of the hereditary anaemias known to produce porotic hyperostosis (Angel 1967: 387; 1971a: 110; 1971b). That found in Africa is attributed to sickle cell anaemia (Steinbock 1976: 235). Its high occurrence in prehistoric Mexican Mayans and southwestern North American Anasazi populations is explained as the result of a maize dependent diet (first suggested by Hooton 1930: 317; later by Schuetz 1979: 62; Ryan 1977; and others). Porotic hyperostosis is not restricted to these geographical areas, but has been identified in skulls from prehistoric Nubia (Carlson *et al.* 1974), Peru (Hrdlička 1914), and Mexico (Hooton 1940). It is also temporally widespread, ranging from a few, extremely rare occurrences in the Palaeolithic (Anderson 1968), to the Mesolithic (Janssens 1970), to the Neolithic (Angel 1967), to the Bronze Age (Cule & Evans 1968), and up to the present (Powell *et al.* 1965). It appears to increase in frequency from Palaeolithic and Mesolithic populations to modern ones.

There has been much debate over the causes of porotic hyperostosis (e.g., as

early as in the 1920's, Williams 1929: 852–5; also sometimes called *cribia orbitalia* or symmetrical osteoporosis). Anaemia of various types is most often cited as the causal agent. In the Mediterranean regions and Africa, causal explanations are relatively easy to posit—thalassaemia or sickle cell anaemia, depending on the geographical location. The above hereditary anaemias do not always provide satisfying explanations, however, as in the case of prehistoric remains from Italy where other factors are postulated (cf. Germana & Ascenzi 1980). Most scientists, especially when describing New World material, ascribe the cranial deformation to iron deficiency anaemia (cf. Huss-Ashmore *et al.* 1982; Moseley 1965; Saul 1973), which is attributed to nutritional deficiencies (e.g., El-Najjar 1977; Moodie 1923*a* and *b*: 126; Peltó & Peltó 1983: 177–82; and others).

Chronic iron deficiency anaemia in hospitalised young children has been documented as causing porotic hyperostosis visible in roentgenograms of eight- to twelve-month old infants (Shahidi & Diamond 1960). Porotic hyperostosis was also visible in roentgenograms of slightly older children with iron deficiency anaemia, specifically in a 17-month-old and 12-year-old, as well as others (Powell *et al.* 1965).

The assumed relationship of dietary stress leading to iron deficiency anaemia producing porotic hyperostosis is a common one. For example, in prehistoric skeletal populations from the Isthmus of Panama which exhibit an increase in the frequency of infections and porotic hyperostosis through time, 'The increase in porotic hyperostosis can be related to the greater dependence on a limited number of dietary resources, primarily maize. The increase in infection rate over time can, in the two agricultural populations, be associated with a greater dependence on fewer dietary resources and a resulting decrease in the nutritional quality of the diet' (Norr 1984: 484).

I propose that other factors—specifically the aggregation of people and sedentism—are responsible for the existence of porotic hyperostosis in many parts of the world. The following concentrates on a case study from the prehistoric southwestern region of the United States. In those areas of the Southwest occupied by Anasazi (other groups will not be examined here), iron deficiency anaemia as the result of a maize-dependent diet has been postulated as the cause of porotic hyperostosis as exhibited in skeletal populations (e.g., El-Najjar n.d.; El-Najjar & Robertson 1976; Kelly 1980; Ryan 1977; and others). These authors support their contention by the fact that iron absorption from maize is poor (e.g., El-Najjar 1977; El-Najjar *et al.* 1976). Studies have focused on the increased incidence of the disease through time, from Archaic hunting and gathering populations to later maize agricultural Anasazi (see Kelley 1980) and are based on the supposition that later Anasazi consumed less meat than did earlier ones (e.g., Ryan 1977). Using data from the Anasazi site of Arroyo Hondo in the Rio Grande River Valley, New Mexico, Palkovich (1984: 436) states that the adoption of intensive agriculture led to marginal 'diets in average years [that] were virtually starvation diets in drought years, which led to the ultimate abandonment of the [Arroyo Hondo] village. Thus, buffering mechanisms of settlement patterns and agricultural strategies could not soften the biological impact of poor diets on the Arroyo Hondo population.'

The following shows that specifically maize, and diet in general, are at most only incidental causes of porotic hyperostosis in the Southwest region. In some areas, deficient diets may exacerbate the occurrence of nutritional iron deficiency anaemia, but an agriculturally dominant and low meat diet was *not* a factor producing the porotic hyperostosis found in southwestern skeletal populations. Instead, I propose that the widespread prevalence of porotic hyperostosis in the Southwest results from population sedentism and aggregation. Aggregation is used here to mean hamlets or larger groupings. This is the common link between such geographically and temporally disparate loci of the disease as prehistoric Egypt, Italy, Peru and the North American arctic, southwest and eastern regions.

The general idea is not necessarily novel (e.g., Armelagos & Dervey 1970; Milner 1983; Scrimshaw *et al.* 1968; Smith *et al.* 1984: 20). That other factors than diet must be involved in producing the porotic hyperostosis found in prehistoric southwestern skeletal populations and its general increase through time has been noted by fewer authors (for exceptions see Martin *et al.* 1985; Weaver 1985). Other anthropologists (e.g., Ryan 1977: 198–200; Kelley 1980: 174–8; Palkovich 1981: 8; Andrews 1972), after acknowledging additional, what they often consider to be secondary factors, believe that the ultimate prime mover behind the increase in porotic hyperostosis through time and between groups in the New World is the result of an increasingly maize-dependent diet. 'It is possible that the death rates are indicative of an overall low level of health caused mainly by malnutrition [at Oak Creek Pueblo, Arizona] . . . Their biggest problem was anemia caused by their dependence on maize' (M. Taylor 1985: 118). In eastern North America, however, some anthropologists have emphasised the synergistic interaction between diet and infectious diseases (Lallo *et al.* 1977: 478–9; also see Mensforth *et al.* 1978: 47–8). Nevertheless, such synergistic interaction models are frequently ignored, or the dearth of meat intake part overemphasised in favour of the more commonly accepted diet model. For example, studies:

suggest fairly consistently that the adoption of farming was accompanied by a decline in the overall quality of nutrition. The clearest indicator of this is provided by the incidence of porotic hyperostosis . . . considered indicative of anemia. Sixteen studies . . . [in the book] note rates of porotic hyperostosis and most conclude that the lesions appear or increase with farming, suggesting that anemia is primarily a disease of agricultural groups. (*Such anemia is most commonly attributed to poor nutrition* but may also be genetic or parasite-related) (Cohen & Armelagos 1984: 587; my emphasis).

The aforementioned diet-oriented explanation for the high infant mortality rate and incidence of porotic hyperostosis at Arroyo Hondo Pueblo, a large fourteenth-century Rio Grande aggregated Anasazi community in the Southwest maintains that 'The malnutrition and related disease conditions themselves may have been the results of infant diet and feeding practices, a general emphasis on high-carbohydrate foods, and/or periodic food shortages caused by local drought' (Palkovich 1980: 47). Others, such as El-Najjar *et al.* (1976); El-Najjar & Robert (1976); Moodie (1923); Pelto & Pelto (1983); Ryan (1977) etc., concur. The belief that Anasazi had a low meat diet is revealed even in articles on

different topics unrelated to Southwestern prehistory. One example of many is, 'The Eskimo with a high-meat diet, exhibited the most frequent type II remodeling, *whereas the Pueblo, with a low-meat diet, showed the least*' (Richman *et al.* 1979: 209; my emphasis), and the authors characterise the Anasazi as 'vegetable-eating Puebloans' (1979: 212). Another example is that in contrast to Plains Indians, 'Southwestern groups continued to develop a specialist strategy dependent on maize horticulture with consequent biological impacts to the populations involved' (Bumsted 1980: 74).

It is important to note that I am aware that diet can be one factor in iron deficiency anaemia. I propose, however, that (1) the prehistoric Anasazi diet in the southwestern region of the United States was nutritionally adequate and (2) other factors associated with sedentary aggregations are the cause of porotic hyperostosis in Anasazi and other populations. Both of these points are assessed below.

How sedentary is sedentism?

How sedentary a group has to be in order to be classified as sedentary is an interesting and debatable question that is further pursued in Kent in press *b*. The question is, however, important enough to be at least cursorily examined here, since sedentism is a major factor for the nondiet-dependent model proposed below. I doubt that many proto- or prehistoric tribal societies spent twelve months a year several hundred decades at a time in one location—that is more common in societies in constrained space, such as on islands, or in areas of high population densities, particularly urban centres.

Even in contemporary sedentary urban Euroamerican society many families take one month or longer vacations and therefore do not spend every month of the year at the same place. It has been suggested that one out of every four North Americans will move, although not necessarily out of a particular city (Chudacoff 1972). In only a five-year period between 1970 and 1975 in the United States, '44 per cent. of the nation's civilian noninstitutional population over 4 years of age resided at a different dwelling' (Berry & Dahmann 1980: 26–8). Of all the moves within this short five-year span, 15 per cent. migrated to locations other than within the community of the original residence (Berry & Dahmann 1980: 28). The sedentary Zulu of South Africa occupy their homesteads for fifteen to thirty years and then move and build a new one; thus each site is rarely if ever occupied for more than one generation (Oswald 1987). I suggest that the factors in operation in settlements occupied for more than a year are similar to those occupied for 100 or more years, and range from constraints on sociopolitical stratification and organisation in terms of the necessity of a leader/arbitrator, to refuse disposal patterning, disease epidemiology, and depending on the size of the aggregation, social integrative mechanisms. It is more productive to view sedentism as a mobility strategy in which a group spends the major part of a year at one locus to which they return after a short, often seasonal, absence (Kent in press *a*; *b*). This would categorise the Anasazi as sedentary.

Anasazi diet

It is commonly assumed that the earlier Archaic people emphasised a hunting and gathering subsistence strategy in contrast to the Anasazi who increasingly through time relied more on agriculture for their subsistence. Reconstructions of Anasazi subsistence are usually based on three assumptions—(1) that the numerical decrease in faunal remains in relation to site population through time indicates proportionately less hunting and consequently less meat consumed; (2) that the larger and later pueblos or villages depleted all potential game within the radius of space inhabitants would be willing to exploit; and (3) that people would rather do without than expend more calories or energy on a resource than they could get by exploiting it.

None of the above assumptions is valid for the prehistoric Southwest for several reasons. First, the fewer faunal remains at many later sites probably indicate more of a change in butchering patterns than in meat consumption. People who have to carry meat a long distance to their settlements often take only those heavy bones needed for a particular purpose, such as raw material to manufacture specific tools. Unnecessary bulk and weight are abandoned at the butchering site (examples are present in !Kung Basarwa [Bushmen] and other hunting-gathering groups). Once animals were hunted out of the immediate area of a sedentary village, people would be forced to travel further to obtain them. This does not mean that they no longer ate much meat; they simply no longer brought the bone refuse back to the site with them. It is most unlikely that people would carry heavy bones to a base camp only to discard them there. So, one should anticipate a change in butchering patterns as a consequence of sedentism and aggregation that does not necessarily reflect a change in diet.

Secondly, contemporary Euroamericans/Europeans who are used to purchasing meat at a local market may feel that a several-day hike to obtain meat was a rare occurrence for prehistoric groups. People without motorised or animal transportation, however, are accustomed to hiking distances we might consider excessive to procure a variety of subsistence, ceremonial and social items. Examples from the Southwest include the Hopi salt trek. Meat can be prepared for long distance transport by drying, but the process results in the removal of bones at the processing locus, which artificially lowers the number of faunal remains at a habitation site. Bones brought back are usually ones used for food, raw material or for a special ceremonial/ritual or social purpose (e.g., the entire carcase from a boy's first major kill; a variable Cordell (1977) did not take into account in her otherwise excellent article on faunal remains and hunting at a Rio Grande Anasazi pueblo).

Thirdly, the Anasazi prehistoric intergroup and interpueblo exchange system could have included meat. This exchange network is known through the extensive trade of ceramics and lithics. Many small to large sites have at least some imported nonlocal pottery, which attests to the prevalence of trade in Anasazi economy.¹ Moreover, recent work from Chaco Canyon reveals a correlation between intra-Anasazi artefact exchange and artiodactyl species frequency at sites. For example, at Pueblo Alto, nonlocal deer replace pronghorn as the primary animal resource at the same time there is an increase in

ceramic tempering materials and Washington Pass chert from the Chuska Mountains where deer would have been more available (Akins 1984).

Analysis of faunal remains from the Gran Quivira pueblos indicates that the Anasazi *did* trade for meat with nomadic hunters and gatherers of the adjacent Plains culture area (Hayes *et al.* 1981: 2). Meat was probably traded for ceramics and other goods, and in fact, Cordell (1985: 18) has noted that 'Rio Grande [Anasazi] glazes [pottery] were widely traded not only in the Rio Grande area itself but also to the adjacent Plains'. People from the northern Pueblos also went to the Plains to hunt for bison. Obsidian from New Mexico sources dated between A.D. 900 and 1400, in addition to Pueblo ceramics, have been recovered at a number of Plains sites (Spielmann 1982: 311-15).

Protohistorically Plains Indians and Navajos traded meat and skins for maize and other agricultural products at 'trade fairs' at Taos and other northern New Mexican pueblos (Gunnerson 1956; etc.). Navajos historically also traded meat and hides at the Hopi pueblos in Arizona (Dyk 1938). That this Pueblo/Plains exchange of corn for meat was also a prehistoric phenomenon is supported by the accounts of the first expeditions by Spanish explorers, such as Coronado, and by slightly later Spaniards who also confirmed the presence of regular Pueblo/Plains exchange (Spielmann 1982: 128). Plains Indians bartered their bison meat, fat, and hides for Pueblo corn, cotton blankets, and ceramics (Spielmann 1982: 128). It is doubtful that pre-horse Plains Indians would have carried entire carcasses to the Pueblo area; instead meat probably was deboned at kill or processing sites, dramatically raising the ratio of meat consumed to bone refuse at the pueblos. The preparation of jerky was observed as occurring at the time of initial butchering (Weltfish 1965 as noted by Spielmann 1982), and bone grease was obtained 'by smashing bones, boiling them in water, and skimming off the fat . . .' (Spielmann 1982: 193). Both procedures artificially lower the ratio of bones recovered to actual meat consumed at sites. Additionally, bison 'robes were observed to have been worn in the winter by all Pecos males . . .' and were used in the interpueblo exchange network (Spielmann 1982: 260). Nonlocal corn has been uncovered at a few Plains archaeological sites, although Spielmann (1982: 296-7) suggests that much corn acquired from the pueblos in exchange for bison products may have been consumed while Plains Indians camped near the Pueblo villages. I suspect that maize was carried back to the Plains, but as archaeologically invisible ground flour, since the cobs are unnecessary bulk and weight for pedestrian Plains peoples.

There is other concurrence that the Anasazi were not as maize dependent as is often indicated by faunal assemblages. Turkeys were available as a meat source prehistorically, as evidenced by 'turkey pens' and consolidated masses of turkey droppings found at sites throughout the Anasazi occupied Southwest (Rohn 1977: 86-7, and others); and are described by early explorers and ethnographers. Early historic sources often mention the procurement of bison meat and the consumption of turkeys. Observed during the Rodriguez expedition in A.D. 1583, 'They [Rio Grande Puebloans] make tortillas and *catoles* with buffalo meat and turkeys, because they have large numbers of the latter. There is not an Indian who does not have his corral in which he keeps his turkeys. Each one holds a flock of one hundred birds' (Hammond & Rey 1927: 26). Moreover, it

has been recorded that protohistorically, both eastern and western Pueblo Indians:

added to their diet by gathering wild, edible plants and by organizing tribal hunts to gather rabbits and other small game. Other delicacies were special species of wood rat, deer, and antelope. When the Spaniards first encountered the Pueblos, most of the villages kept vast flocks of turkeys, both for food and feathers (Schroeder and Matson 1965: 115). Periodic trips to the Plains country to hunt buffalo are also well-remembered activities among old Pueblo residents. It is likely, however, that buffalo meat and skins were obtained primarily by trade or gift exchange with friendly nomadic tribes (Dozier 1970: 129).

Butchering marks on *Canis* spp. (probably dog) uncovered from a small Pueblo II, circa A.D. 1030–1050, habitation site in the Mesa Verde Anasazi region suggests an additional available meat source not usually recognised (Kent in preparation). At the same site 13.55 per cent. of the faunal assemblage consisted of turkey bones, rabbits made up 44.06 per cent., rodents 26.01 per cent. and dogs 2.26 per cent. Large and medium sized mammals made up only 11.86 per cent. of the assemblage (Kent in preparation). When dog is identified at a site, their bones are often not carefully inspected for butchering marks since it is assumed that people would not eat 'man's best friend'. Even when such marks are noted, they are not always included in the list of domesticated food exploited, although this is changing (see Emslie 1985). Dogs, then, might add another locally available meat resource not commonly acknowledged.

Even in those areas where there was prehistorically a relatively high population density, meat was probably still available. Perhaps turkey, rabbit, wood rat and other small animals were the meat staples of the Anasazi diet, with deer, bison, various birds, pronghorn antelope, dog and other animals being consumed less frequently. In fact, consumption of meat sources typically considered inedible by many Euroamerican archaeologists but with high nutrition, such as grasshoppers, are indicated by coprolite analysis. For example, coprolites from Anasazi sites indicate that meat was consumed along with bone cakes, rodents and grasshoppers (Fry & Hall 1975: 95). Furthermore, 71 per cent. of the coprolites identified from Pueblo Alto in Chaco Canyon contain bone fragments (Clary 1984). Out of thirty-five samples, 1,305 bone fragments were counted and most were deer mouse, prairie dog or cottontail rabbit remains (Clary 1984: 269). That 71 per cent. of the coprolites from one Anasazi village contain bone fragments from species often considered intrusive to sites simply does *not* support the usual portrayal of the Anasazi diet as a primarily vegetarian one.

Agricultural fields actually *promote* the abundance of certain animal species and hunting them is sometimes referred to as 'garden hunting'. In addition, approximately 67 to 76 per cent. of the live weight of small mammals, such as rodents, is edible meat (Stahl 1982: 823–7). Unfortunately, small animal bones are less visible archaeologically and more subject to carnivore activity and erosional processes than large bones (Binford & Bertram 1977). Archaeologists have often discounted rodent bones as intrusive because rodents are not an animal Euroamericans prefer to eat, unless they are starving. Even today, such animals are often considered to be 'starvation food', since it is assumed that

every good Anasazi would prefer the meat from larger animals if available, an unwarranted assumption.

Some archaeologists have argued that the low occurrence of faunal remains coupled with the small emphasis on hunting described in Pueblo ethnographies indicates a paucity of hunting, and by extension, of meat consumed by pre- and protohistoric Pueblos. There are a number of problems with this assertion, the least of which are the inherent problems of using the direct historical approach of ethnographic analogy, as detailed in Kent 1987. But even more problematical is that a more thorough study of Pueblo ethnographies reveals that the analogue is incorrect ethnographically and Pueblos did hunt substantially more than is commonly thought. There are tangible reasons for the dearth of bones at historic sites. For example, the mythology of eastern Pueblos strongly suggests that hunting formerly played a much greater part in their economy than is often recognised, the scarcity of faunal remains being the result of the modern Pueblo practice of depositing bones at shrines usually located outside the village (Ortiz 1969: 176). Modern pueblos still today perform the 'buffalo' dance in which bison and other animals are symbolically hunted by dancers. Furthermore, the Spaniards caused the historic decline in Pueblo subsistence hunting by introducing new crops, better agricultural technology and animal husbandry that depleted the fauna near settlements, as well as more efficient weapons to hunt animals, an increase in the area's population, new settlements and appropriation of range land (Ortiz 1969: 176-7).

The above discussion of Anasazi diet negates many anthropologists' assumptions that the Anasazi were singly dependent upon a maize diet. This is in contrast to, for example, El-Najjar's (1977: 329) assertion that 'Heavy dependence on a single food item, such as maize, may have been responsible for the high incidence of [porotic hyperostosis] in the New World. . . . The nutritional properties of maize together with maize processing techniques are the two main factors responsible for porotic hyperostosis in groups whose diet consisted primarily of maize.' While this may be true of a hypothetical population with a heavy dependence on a single food item, especially maize, it is unlikely that such populations actually existed very often prehistorically. Such populations are known historically due to the presence of Europeans or Euroamericans, overcrowding and precipitous population increases partly a result of improved health care, as well as over-exploitation of the land, etc. For even if the Anasazi did not eat much meat (and the data suggest otherwise), maize probably did not constitute the 75 per cent. of their diet that has been claimed. Just one example can be seen in El-Najjar and Robertson's (1976: 143) contention that 'Among inhabitants of environments similar to that of Canyon de Chelly . . . maize constituted over 75 per cent. of the diet'. Beans and squash, as well as other plants, were also cultivated.

Twenty identified plant taxa have been recovered to date [from coprolites at a large Anasazi pueblo] including the following common names: amaranth/pigweed seed, cactus family epidermis, chenopod/goosefoot seed, beeweed seed, squash seed and tissues, horsebrush stem, cotton seed and fiber, grass family seed, sunflower seed, pea family seed, bean fragments, groundcherry seed, pine nut fragments, pine resin, purseland seed, squawbush seed, dropseed, and corn seed and tissue (Fry & Hall 1975: 95; see also Williams-Dean & Bryant 1975).

Whereas maize has a relatively low iron absorption rate (El-Najjar 1977), other Anasazi crops have higher iron absorption values. Maize has an iron absorption value in a normal average male of only 0.9; however, black beans have an absorption rate of 12, which is higher than that of either fish (2.8) or even veal (9.2; Layrisse *et al.* 1969: 441). These values change when the items are consumed by an individual with iron deficiency anaemia: 'Normal subjects absorb 5 to 10% of dietary iron, compared with about 20% in iron-deficient patients' (Lanzkowsky 1980: 47). Also important is the fact that plant and animal food, if eaten in combination, significantly increases the absorption of iron from vegetable sources, perhaps because '... several of the amino acids, particularly cystine, lysine, and histidine, have been found to be effective in increasing the absorption of iron' (Lanzkowsky 1980: 46; also see Hoffbrand 1981: 38-9). The consumption of maize, beans and squash in the Southwest, augmented by various wild plants and meat from small game and occasional large game, probably produced a nutritious diet adequate in iron.

Sedentism and aggregation: an alternative model

If not a consequence of diet, then other causal agent(s) must account for the incidence of porotic hyperostosis in the Southwest. In contrast to the diet model, I suggest that the increase in porotic hyperostosis can be better accounted for by the increase in sedentism and aggregation (i.e., the occupation of hamlets or larger settlements). Through time in the Southwest, as well as elsewhere, reliance on horticulture increased. Also through time, however, settlements became more sedentary (using the definition presented earlier) and aggregated. In other words, there is a correlation between an increasing agriculturally oriented Anasazi subsistence strategy and an increasing sedentary aggregated settlement pattern as compared to the earlier Archaic. I have attempted to demonstrate that Anasazi horticulturalists did not have chronic iron deficiency anaemia resulting in porotic hyperostosis as a result of their diet. The following will demonstrate that sedentary and aggregated Anasazi did have chronic anaemia as a result of their demography. At different times in different branches of the Anasazi, large sedentary villages of several hundred rooms were occupied (e.g., Pueblo II Chaco Anasazi and Pueblo III Mesa Verde Anasazi; see Cordell 1985). The majority of skeletons discussed below are from the time periods of the large villages.

From a sample of eighty-four Anasazi skulls, ranging in time from early Basketmaker to Pueblo III, 24.7 per cent. exhibited porotic hyperostosis, most being in immature skeletons (Zaino 1967: 42). It is important that infants from Alkali Ridge, Utah, and elsewhere had active cases of porotic hyperostosis, whereas adults had indications of healed manifestations of the condition (Brues 1946: 328). Of 539 crania from various Anasazi large aggregated sites throughout the Southwest, 34.3 per cent. exhibited evidence of porotic hyperostosis (El-Najjar *et al.* 1975). Children, as is typical, had a higher incidence than adults. Anasazi skeletons from Chaco Canyon, where very large sites occurred, had the highest percentage of the disease (71.8 per cent. of 32 individuals) followed by

Inscription House (54.2 per cent. of 24 individuals), Gran Quivira (15.3 per cent. of 177 individuals) and Navajo Reservoir where there are no large villages (13.0 per cent. of 92 individuals (El-Najjar *et al.* 1976: 481–5). The earlier Basketmaker period skeletons from Canyon de Chelly had 49.3 per cent. of 136 individuals affected whereas the later Pueblo period skeletons had 55.1 per cent. of 78 individuals affected (El-Najjar *et al.* 1975: 922–3).

The difference between early less horticulturally oriented Basketmaker and later more horticulturally oriented Pueblo skeletons is only 5.8 per cent., casting some doubt on the magnitude of difference in porotic hyperostosis between a lesser and greater emphasis on a horticultural subsistence orientation, but not on the effects of aggregation and sedentism since some Basketmaker villages were large. It is also important to take sample size into account when viewing these figures; for instance, disparate sample sizes from a poorly known or unknown universe were used to calculate the aforementioned percentages. This does not however deny the fact that porotic hyperostosis resulted from a fairly common malady among prehistoric groups; nor does it mean that one should simply discard the data for fear of invalid conclusions resulting from potential sampling bias. It should, however, make one somewhat cautious, especially since more complete excavations yielded lower percentages of the affliction, as indicated below.

Eighteen Pueblo III burials at Site 34 in Soda Canyon at Mesa Verde, Colorado, had porotic hyperostosis, in addition to a few skeletons with arthritic exostoses, cysts and mastoid abscesses together comprising one half of the skeletons recovered (Reed 1965: 41). The Wetherill Mesa Project, Colorado, uncovered an additional 179 partial and complete burials with only six infant skulls from all three Pueblo periods displaying any evidence of porotic hyperostosis, in addition to five definite and two probable cases of infections (mostly osteomyelitis) (Miles 1966; 1975). Of the 581 skeletons uncovered at the protohistoric/early historic Anasazi site of Pecos village, 3.27 per cent. had porotic hyperostosis (Hooton 1930: 320).

Other evidence of prehistoric health problems includes a human fluke infection (digenetic trematoda of unknown genus) discovered in coprolites from a late period Anasazi site (A.D. 1250–1300) in Glen Canyon, Utah (Moore *et al.* 1974: 115–18). Many flukes that invade humans can cause diarrhoea, as well as malnutrition by robbing their host's digested food, although species identification of the fluke is needed before its potential effects can be ascertained (Brown 1975: 209–54). Other evidence of parasites in the prehistoric Southwest includes eggs from pinworm (*Enterobius vermicularis*) found in fecal material at sites in Colorado (Samuels 1965: 175–8), and Utah, and Arizona (Fry & Hall 1975: 94). The fact that remains of parasites have been recovered from prehistoric sites indicates not only their presence, but their prevalence as well since the statistical odds of finding coprolites with parasites analysed by people competent to identify them is actually very low.

The previous section elucidated the superficial correlation between Anasazi maize horticulture—which actually always included beans, squash, some now wild plants and meat—and porotic hyperostosis. The misleading correlation between agriculture and porotic hyperostosis is spurious, and other factors,

specifically sedentism and aggregation, also tend to go along with increases in an horticultural subsistence strategy. Because it is unlikely that the Anasazi ever subsisted primarily on maize, porotic hyperostosis is more likely the result of the relationship between anaemia and sedentary aggregations, than between anaemia and diet. The ramifications of sedentary village life include increased bacterial, viral and parasitic infections due to poor sanitary conditions and contact with potential hosts of carriers of infections. Those prehistoric south-western groups with lower incidences of porotic hyperostosis labelled 'non-maize dependent groups' by El-Najjar (1977), or in other words, less agriculturally dependent people such as the Navajo Reservoir area groups, were also less sedentary and lived in smaller aggregations than the 'maize-dependent groups' (the latter being inhabitants of Chaco Canyon, Canyon de Chelly, and Inscription House sites).

Iron deficiency anaemia: causes and consequences

What then caused the incidence of porotic hyperostosis, and by implication iron deficiency anaemia, among the Anasazi? Iron deficiency anaemia is common in humans today and, judging by the archaeological record, was common in the past. Currently iron deficiency anaemia is widespread throughout the world. The impact of diarrhoeal diseases on an individual's health is not always recognised, since European/Euroamericans rarely have the frequency or severity of symptoms as have many non-Western peoples. Because of Western medicine (and sanitation standards), the effects of diarrhoeal diseases often can be lessened or eliminated before they weaken or kill. Such medicine is scarce in some Third World nations and was not available prehistorically, although native medicine no doubt alleviated some symptoms. One can gain an appreciation of the prevalence and severity of these diseases that are often thought to be relatively benign in contemporary Western societies by examining the health problems rampant around the world. For example, a minimum of 35 per cent. of the deaths of Brazilian infants in 1958 was the result of diarrhoeal diseases (Penido 1959: 368). No less impressive is the fact that diarrhoeal diseases account for one half of all hospital admissions for children under five years of age in Honduras (Kendall 1981: 1). In Venezuela alone, almost 84 per cent. of all deaths from gastroenteritis occurred before a child was two years old and between 1950 and 1954 diarrhoeal diseases accounted for 60 per cent. of the deaths in rural towns with populations of fewer than 5,000 people (Curiel & de Ochoa 1959: 353-4). One study states that 'diarrhea, as a cause of death, almost equals all the other causes combined, even if respiratory tract infections are included. The World Health Organization . . . estimates about 750 million episodes of acute infectious diarrhea every year. . . . Recent WHO figures for worldwide mortality for young children with diarrheal diseases indicate 4.3 million annual deaths, excluding China' (Flewett 1984: 343).

Iron deficiency anaemia is one of the most prevalent modern deficiency diseases and its incidence varies in different countries from 30 to 95 per cent. (Heilmeyer & Harwerth 1970: 381). In Europe it is present in 25 per cent. of post-pubertal females in Sweden (Halberg 1970: 457), in 15 per cent. of females

in the United Kingdom (Kilpatrick 1970: 443), in 13.9 per cent. of people in Germany (Seibold 1970: 438) and in the late 1970's, in 17-44 per cent. of North American children between six months and three years of age (Lanzkowsky 1980: 57). A vegetarian diet, the cause usually suggested for historic cases of anaemia in Europe and prehistoric cases of anaemia in the New World, cannot alone account for these impressive statistics. Both aggregation and sedentism, however, *can* explain present and past incidences of anaemia.

Most of the archaeological examples of porotic hyperostosis are from children under the age of five, which is consistent with the age of the most severe manifestations of bacterial, viral and parasitic diseases. The factors involved will be detailed to demonstrate their relationship in fostering iron deficiency anaemia as a consequence of people becoming more sedentary and aggregated, a setting which creates more person-to-person contact and greater opportunity for the transmission of gastrointestinal diarrhoea-producing diseases.

Diet

The most often cited nutritional cause of iron deficiency anaemia is a low iron or meat intake. In those parts of the world where meat is scarce, nutritional deficiencies play an important role in causing and maintaining high levels of anaemia (Chandra 1979; Baker 1978; Hummert 1983); the prehistoric Southwest was not, however, such an area. In infancy and early childhood, when milk is the major portion of the diet, it was and is still often thought that iron deficiency anaemia is a consequence of the low iron content of mother's or cow's milk (Betke 1970; Palkovich 1984, etc.). Nonetheless, prolonged lactation by itself does not often result in anaemia, as several recent studies have shown, despite common beliefs otherwise. While I am not suggesting that a completely iron-deficient diet will not cause iron deficiency anaemia, I am suggesting that the prevalence and severity of anaemia that occurs in some young children does so despite a diet adequate in iron and meat.

The protective effect of breast-feeding against infections has been recognised clinically for decades, particularly in relation to diarrhoeal disease in conditions of poor hygiene (Jelliffe & Jelliffe 1978: 5). Mother's milk provides protection to vulnerable infants from *Escherichia coli*, a diarrhoea-producing bacterium which is virtually nonexistent in breast-fed young children in contrast to formula-fed children (Gryboski & Walker 1983: 547). Furthermore, breast-fed infants, particularly up to nine months of age, have a significantly reduced risk of respiratory infection, including pneumonia, than have nonbreast-fed infants (Forman *et al.* 1984). Studies also indicate that human milk protects against *Vibrio cholerae*, *Shigella*, *Salmonella*, rotavirus, and toxins A and B of *Clostridium difficile* (Kim *et al.* 1984; Hjelt *et al.* 1985; Ryder *et al.* 1985).

Host resistance factors in human breast-milk properties act mainly within the intestine and the active anti-infective property of human milk is both humoral and cellular (Jelliffe & Jelliffe 1978: 85). Lactoferrin, lysozyme, interferon and other antibacterial and antiviral agents occur in human milk (Raiha 1985) and new research shows that exclusively breast-fed infants are generally more healthy than nonbreast-fed ones (cf. Forman *et al.* 1984). In comparing fatal

versus nonfatal cases of acute diarrhoea in thirty-two Cuna Indian children under the age of five from islands off the coast of Panama, all nonfatal cases were exclusively breast-fed at least during the first four months of life in contrast to the fatal cases of diarrhoea in which only 62 per cent. were exclusively breast-fed for the first four months, 25 per cent. who were never breast fed, and 13 per cent. who were intermittently breast-fed (Ryder *et al.* 1985: 607). Breast feeding reduces the severity of the clinical manifestations of specific viral agents in cases where it does not actually prevent it (Duffy *et al.* 1986). Most interesting in terms of this study is the suggestion that because 'shigellosis continues to account for substantial morbidity and mortality in children . . . the results [of our study] supports prolonged breastfeeding . . . [up to 35 months]' (Clemens *et al.* 1986).

I suggest that the correlation between diet (mother's milk) and infant anaemia is actually spurious. Instead, I suggest the anaemia noted in some breast-fed infants is the consequence of maternally transmitted viral, bacterial and/or parasitic infections. Enterotoxigenic strains of staphylococci have been found in breast milk of healthy mothers whose children have diarrhoea (Adekeye & Adesiyun 1984: 531). 'The fact that 71.3% of breast milk samples from 251 healthy nursing mothers contained [*Staphylococcus aureus*] is worrying, especially as 48% of the isolates produced enterotoxins. The incidence of enterotoxigenicity may even be higher, as we only looked for SEA, B and C' (Adekeye & Adesiyun 1985: 536). The more frequent such infections are present in the general population, as I suggest they are in sedentary aggregations, the more likely the transmission of infectious agents to an infant through mother's milk. Authors from around the world have recently noted similar findings concerning the transmission of disease vectors through lactation. This seriously questions the role of diet in causing anaemia in breast-fed infants, and instead implicates bacterial and viral agents passed on to a child through mother's milk. Anaemia, in other words, is not solely dependent upon how much iron is ingested, but on how much is absorbed by the body. These important factors are discussed below.

Infections and traumas

Nongastrointestinal infections or inflammatory states of various types and traumas such as fractures are also potential causes of anaemia (referred to as anaemia of chronic disease). By 1952 it was known that anaemia is most likely to develop from (1) severe rather than mild infections (i.e., 10 per cent. of patients in one study with moderately severe chronic infections developed anaemia versus 40 per cent. of those with severe infections), (2) infections lasting a month or longer, and (3) infections in infants and young children (Cartwright & Wintrobe 1952: 168). Perhaps of more significance for the present study is the fact that anaemia developed in infants after a series of infections, *even though their diets were unquestionably adequate* (May *et al.* 1952). The type of infection present is not as important in causing anaemia as its duration and severity (see de Gruchy 1978: 197-9; Reizenstein 1983: 92-4, the latter includes a description of the

severe anaemia resulting from pulmonary tuberculosis and rheumatoid arthritis). This is particularly interesting when one considers the frequency of rheumatoid arthritis, osteomyelitis, tuberculosis and so on, in prehistoric sedentary populations (cf. as identified by Hooton 1930; 1940; Fry 1976; and others). Even with massive intravenous therapy, and adequate dietary iron intake, anaemia can result as a consequence of rheumatoid arthritis; although the link between such chronic inflammatory diseases and anaemia is not yet known, it may be related to immune mechanisms (Hurley 1983: 132; Reizenstein 1983: 47; 70). Arthritis, though not always rheumatoid, is perhaps the single most common pathology other than dental ones found in prehistoric southwestern populations.²

Several factors implicated in the pathogenesis of the anaemia associated with chronic diseases include (1) a decreased production of red blood cells resulting from relative erythropoietic failure and a defect of iron mobilisation from the reticuloendothelial tissue, and (2) a shorter red blood cell life survival from 120 days to only 60 to 90 days (Reizenstein 1983: 42–4 and Zucker 1980: 94). In other words, all the variables are present for healthy haemoglobin/haematocrit levels but are not utilised efficiently by people with chronic infections or inflammations. In addition to these changes as a consequence of chronic infections or other inflammatory processes, chronic anaemia can cause changes in the stomach—gastritis to gastric atrophy—in individuals of all ages. When in the small bowels of children, chronic anaemia can result in occult blood loss in the stools among other changes (see Jacobs & Worwood 1982: 176). This can perpetuate the anaemia or increase its severity.

Also relevant to prehistoric populations is the fact that trauma such as fractures, contusions, or extensive lacerations can produce short term anaemia during the first week or two following an accident, potentially aggravating any underlying problems. This anaemia is not the result of blood loss, infection or hemolysis, but is, instead, the result of a disturbance in haemoglobin synthesis. In fact, 'Slight reductions [in the serum iron concentration] also are seen even after the most minimal trauma such as a small operation under local anaesthesia and a simple smallpox revaccination . . .' (Reizenstein 1983: 72). As Scrimshaw (1968: 708) has noted, 'Any cause of chronic bleeding will contribute to the development of iron-deficiency anemia'.

Diarrhoeal and other gastrointestinal diseases

The prevalence of a number of diarrhoeal and gastrointestinal diseases increase in response to sedentism and aggregation. Diarrhoeal diseases can cause iron deficiency anaemia in two ways—by passing the food through the intestines too rapidly for most of the nutrients to be absorbed, or by actual blood loss, common to such diseases. In fact, nondiarrhoeal infections cause overt malnutrition as a result of a possible increase in 'resting metabolic expenditure during infection stress rather than the early fall in metabolic rate that characterizes starvation or underfeeding' and this causes a more rapid and severe weight loss than does starvation alone (Duggan et al. 1986: 65). Specifically with

gastrointestinal infections, a sequence of hormonal, metabolic and immunologic events create a precipitous nutritional cost (Santos 1986). Fever, anorexia, vomiting and negative nitrogen catabolism associated with diarrhoea result in food withdrawal in order to rest the small intestine resulting in a 20 to 60 per cent. or more deprivation of a child's normal caloric consumption (Kumate & Isibasi 1986: 25).

In general, both bacterial and viral diarrhoea-causing infections have a higher frequency in children than adults and the symptoms are more severe and malabsorption more prevalent in children, especially the younger ones (Ackers 1982: 422; Wilson *et al.* 1981: 131-2). A study from Scotland revealed that the majority (83 per cent.) of rotavirus infections appear in children under five (Cash *et al.* 1986). Specifically in the cases of rotavirus and enterotoxigenic *Escherichia coli*, two of the most common ailments, small children appear to lose a greater proportion of their total fluid volume during diarrhoea episodes and as a consequence have a higher frequency of severe dehydration and death (Black *et al.* 1984: 83). The average diarrhoeal stool loss rate in infants is more than twice that in adults with enterotoxigenic *Escherichia coli* (Black *et al.* 1984: 87). A similar pattern has been associated with coronaviruslike particles (Rettig & Atshuler 1985).

Weanling diarrhoea can cause anaemia and death and is linked to weaning a child from mother's milk. For example, from 1955 to 1959 in India, 22 per cent. of all deaths during infants' first year of life and 9 per cent. of all deaths of children two to three years old were attributed to weanling diarrhoea (Gordon *et al.* 1963: 371). Some of these deaths might not be associated with diet or weaning but were caused by increased contact with ill or asymptomatic carriers of infections as a child became older and was in contact with more people and/or suffered the loss of some of the immunity afforded by mother's milk.

Whatever the actual cause, abnormal small intestinal lesions correlated with acute diarrhoea, especially during the first year of life, may contribute to the perpetuation of the diarrhoea and be responsible for the lactase deficiency and lactose intolerance associated with such diarrhoea (Fagundes-Neto *et al.* 1984: 510, 514-15). Another complication of acute gastroenteritis and protracted diarrhoea is monosaccharide intolerance associated with rotaviral infections but also present in their absence, perhaps when multifactorial protracted diarrhoea results in small intestinal mucosal damage (Manuel *et al.* 1984: 44; also see Brandt, *et al.* 1985: 437-443). This condition in infants varies in duration from 2.5 to 70 days in at least one study (Manuel *et al.* 1984) and contributes to the length and severity of a diarrhoeal disease, potentially making an infant vulnerable to other diseases present in the community.

Important is that many of the bacterial, viral and parasitic infections that cause diarrhoea actually increase as sanitation decreases and are rampant in non-Western populations settled in sedentary aggregated groups which lack the amenities common in Euroamerican/European societies. Sanitation decreases in such aggregations, usually as a result of poor and contaminated water supplies, as well as being associated with certain practices. For instance, while conducting participant-observation fieldwork among Navajos in a remote part of the Reservation (Kent 1984), I observed unrefrigerated and unprocessed meat

cooked and eaten in midsummer as long as two weeks after a sheep had been butchered. This custom no doubt enhances the chances of contracting a diarrhoeal disease. Drinking and washing water was hauled from a tribal well several miles from camps and stored in open barrels where disease-carrying insects and dirt would gather. Outhouses were used at some camps, but were often not present at all, and when absent, an arroyo or gully was used which was a potential contaminant of water sources over a wide area. Another example was the use of the same metal wash tub to wash dirty nappies and to clean dishes in cold water (cf. Kent 1984). Obviously, this presented an excellent vehicle for the spread of disease, especially since the infant and I both had diarrhoeal diseases—mine being diagnosed as a *Shigella* infection—at the time. In developing nations, children under the age of five years are ill on an average of 34.2 to 35.2 per cent. of the time during an one and a half year period according to one study (McAuliffe *et al.* 1985).

Bacterial and viral diseases

Common bacterial diarrhoeal diseases resulting in gastroenteritis are caused by *Shigella*, *Salmonella* and *Escherichia coli* bacteria (Wadstrom 1978; Levine & Hornick 1981: 254, 256). All can be responsible for mild to severe diarrhoea and all procreate in conditions of sedentary aggregations. Susceptibility of young children to these bacteria corresponds to the peak incidence of porotic hyperostosis found in prehistoric infants and young children's skeletons.

Most bacterial diseases can be transmitted to humans by rabbits, ticks, deer flies, dogs, fleas and other animals. This is particularly significant when one realises that domesticated dogs and turkeys were kept by the Anasazi and that the later archaeological record indicates an increased reliance on rabbits and other small animals for food. These bacterial diseases are aggravated by the sedentism and aggregation of people because of an increase in potential vectors present near or in the habitations. Diarrhoea, vomiting, and nausea characteristic of bacterial infections can occur through the ingestion of contaminated meat or water, resulting in primary lesions in the gastrointestinal tract (Cluff 1980: 661). For example, tularemia (rabbit fever) can be transmitted to humans from infected rabbits, dogs, flies, and other animals with which the Anasazi were likely in contact (Cluff 1980: 661).

Viral gastroenteritis is the second leading cause of illness in the United States, and no doubt caused illness among the prehistoric Anasazi. Currently paraviruses are responsible for diarrhoeal diseases in adults and older children and rotavirus in young children. Rotavirus agents are the underlying causes of diarrhoea in many younger children and infants because they have fewer antibodies than adults to protect them against the virus (Lerner 1980: 814). The link between viral infections and haemolytic anaemia is well known, if poorly understood. The relationship has been suggested to be connected in some way to the autoimmune system as mentioned earlier (Lascari 1984: 1). Rotavirus has been diagnosed in children with diarrhoea, vomiting, and in some cases limited gastrointestinal bleeding (Clemens *et al.* 1983; Paniker *et al.* 1982: 123–6; Stintzing *et al.* 1981: 67–71). In summary:

In developing countries, where sanitation is generally inadequate, young children develop multiple episodes of diarrhea (often four to eight per year) during the first 2 to 3 years of life. . . . Most of these diarrheal episodes are mild, but some are life-threatening. All probably contribute negatively to the nutritional development of the child (Carpenter & Sack 1981: 213).

Parasitic infections. The actual prevalence of parasites in the United States is quite variable and in the early 1900's, their numbers ranged from 93.9 per cent. of 83 Arapahoe Native Americans living at a mission school to 77.1 per cent. of 83 children in an industrial school, to 71.0 per cent. of 42 orphans in Oregon to virtually nonexistent in some Euroamerican populations (Owen *et al.* 1934: 913-15). A number of protozoa can produce acute diarrhoea.³ Amoebic dysentery (*Entamoeba histolytica*) for example causes symptoms from chronic mild diarrhoea to fulminant dysentery with bloody stools and is still found on Native American Reservations and anywhere crowding and poor sanitation exist (Brown & Neva 1983: 30-3; Plorde 1980: 863). Helminth parasites not only rob the body of essential nutrients, such as iron, but also can cause occult blood loss and/or diarrhoea. Finally, fungal infections and chronic nonspecific diarrhoea syndrome or irritable colon of childhood can also cause chronic diarrhoea (Gryboski & Walker 1983: 573-7), potentially leading to anaemia.

The epidemiology of chronic gastrointestinal diseases

Relatively large amounts of both rotavirus and Norwalk virus diarrhoea-producing agents have been detected in untreated and treated, including chlorinated, drinking water in developed and undeveloped countries (Cliver 1984; Herbert 1985; Zohar *et al.* 1984; Goddard & Sellwood 1984; Gerba 1984). How the viruses enter water sources is not fully understood, although many probably do so from infected faeces located near groundwater. A study from Alaska indicates that viruses can contaminate sources as far as 317 km downstream and live for at least 7.1 days as they travel that distance (Melnick 1984: 6-7). In addition, infected wild animals, such as ducks with viral infections, can contaminate water that is then used by humans, beginning a cycle of chronic human infection (Melnick 1984: 14). Blue herons, beavers, and muskrats have been implicated in the spread of *Giardia* infections (Georgi *et al.* 1986) and chickens in the spread of *Campylobacter* (Harris *et al.* 1986). Viruses of various types have been identified in a wide range of waterfowl and other birds in North America (Hinshaw *et al.* 1985; Sacks *et al.* 1986), in pigs and turkeys in France (Aymard *et al.* 1985), and in wild opossums in the Amazon region (Linhares *et al.* 1986). There is additionally some evidence of certain rotaviral strains which may be transmitted by a respiratory route (Santosham *et al.* 1985).

Chronic infections start through contaminated sources, but are maintained, even in a small hamlet or aggregation, by the rapid evolution of some micro-organisms. These new strains can infect even those with antibodies against the original strain, as well as infecting those without body defences to resist. 'The high mutation rate of RNA viruses means that mutants could possibly emerge during a single infection of an individual and, given a selective advantage, such

mutants could predominate in the progeny virus' (Oxford 1985: 1; see also Konno *et al.* 1984; Nicolas *et al.* 1984). A number of RNA viruses including influenza can have a rate of antigenic mutants in virus pools of one in ten (Oxford 1985).

The tendency for rapid evolution even within a single epidemic negates some of the immunity afforded by a previous exposure and infection by rotavirus and Norwalk virus (cf. Oxford 1985; Ryder *et al.* 1985). Protection by immune factors produced against rotavirus and Norwalk viral neonatal infections may not prevent reinfection from the same virus but often can lessen the severity of the disease the second time around (Ryder *et al.* 1985: 103). Even so, a study of 369 children under the age of 4.17 years (50 months) in Bangladesh showed 'that 46% of the children developed NW [Norwalk virus] infection over a 12-month surveillance period. Adjustment of our [own] seven-month surveillance rates to an annual basis for comparison reveals that 63% of San Blas Cuna Indian children [from Panama] less than 48 months old had NW [Norwalk viral] infection' (Ryder *et al.* 1985: 104). Although perhaps immune to reinfection from the same identical strain, weakened individuals recovering from an infection may be more vulnerable to different strains evolved during a single epidemic.

An epidemiological model for the transmission and perpetuation of rotavirus has been proposed by Holdaway *et al.* (1985) wherein those over fifty years of age act as a reservoir of rotavirus in a community while rarely suffering gastroenteritis symptoms themselves. The post-fifty-years-old group then passes viruses to, and causes new infections in, children under ten years of age. They subsequently spread the virus to young adults between twenty and twenty-nine years old, who are involved in the care of the children. Support for this model comes from observations that the over fifty-years-old group had a higher than expected IgG antibody response at all times of the year while at the same time having lower than expected levels of IgM in contrast to the one to ten and twenty to twenty-nine-year-old groups, both of which had high IgM levels (Holdaway *et al.* 1985: 396). This is because IgM antibodies are the first produced to an antigenic exposure, such as a bacterium, and therefore a higher titer implies an acute response to the antigen, while an IgG antibody is produced later in the sequence of immune response.

All this is complicated by the presence of disease carriers in the most vulnerable age group, those under the age of five, in addition to the older age group reservoir. According to Champsaur *et al.* (1984b: 682), during the time when rotaviral infection and disease are most common—that is, in the one to 24 months of age period—two out of ten children hospitalised had the actual rotaviral disease (with serological response and diarrhoea), one had an asymptomatic rotaviral infection (with serological response and no diarrhoea), two were virus carriers (no serological response), and five were not affected by rotavirus (Payne 1984; also see Champsaur *et al.* 1984a). Faecal specimens from 1,841 children under the age of two without gastrointestinal symptoms yielded almost the same percentages of salmonellae and shigellae as children with diarrhoea (Ho & Wong 1985: 55). It is by maintaining a reservoir of disease, with organisms that can mutate at rapid rates which are potentially able to reinfect a

'compromised' host, that a cycle of infections can be initiated that would be difficult to break without modern medicine, as is the case in developing countries and was the case prehistorically.

Recent studies strongly indicate that primary viral infections increase host susceptibility to secondary microbial invasion and increase susceptibility to opportunistic fungal and protozoal pathogens as well (Mills 1984: 469-73). Although most studies have been restricted to pulmonary infections, they provide a model for probable gastrointestinal viral-bacterial synergistic interaction. One manner in which this interaction occurs is by viral depression of normal neutrophil and macrophage activity that would otherwise destroy bacteria (Babiuk 1984a: 43; 1984b). Viral infections can also impair chemotactic factors which are critical to host defence in early bacterial invasion (Mills 1984: 473; Babiuk 1984a: 436-7). Viral infections additionally appear to enhance adherence and colonisation of both gram-positive and gram-negative bacteria by altering cell surface, necessary for gastrointestinal tract infections which then allows bacteria to propagate within the intestines rather than being flushed harmlessly through the system. This is accomplished by reducing the levels of fibronectin on the surface of epithelial cells (by killing mature cells and increasing the number of immature ones with less fibronectin) and increasing cell surface proteases, thereby favouring bacterial adherence and colonisation (Babiuk 1984a and b).

The link between primary viral and secondary bacterial infections can be clearly demonstrated in the respiratory tract. Bacterial adherence to epithelial cells, at least in the respiratory tract, is required for bacterial infections but requires the penetration of the mucous layer that covers epithelial cells (Babiuk 1984a and b; Mills 1984). 'Viral infections may be just the trigger to increase or decrease the quantity as well as the quality of this mucous layer and cause the epithelial damage required for the primary [bacterial] colonization' (Babiuk 1984a: 434). Suppressed bactericidal activity from phagocytic cell dysfunction helps create this situation. Further virus-induced immunosuppression includes the lymphocytes and specifically cytotoxic T (suppressor) cells (see Babiuk 1984a: 432-8 for details). A similar course of events probably exists in the interaction between viruses and bacteria in the gastrointestinal system, but has not yet been as well documented.

Discussion

An epidemiological model can thus be advanced which better accounts for the incidence of porotic hyperostosis among the Anasazi than the more common but simplistic model of a low meat diet. A cycle of chronic viral, bacterial and parasitic infections begins with the lack of sanitation that often accompanies sedentary aggregations. In these settings, waste accumulates and is potentially introduced into a community via contaminated groundwater used for drinking and washing and/or contaminated animals such as dogs, rats, flies and toddlers. Diarrhoeal-producing viral infections are perpetuated within a sedentary aggregated community by contact with infected asymptomatic carriers who act as a

reservoir for maintaining disease within the community. Compromised individuals, particularly those under the age of five who are most vulnerable and most severely affected by diarrhoeal diseases, can be reinfected by a mutant strain during the same epidemic, as well as be predisposed to bacterial and parasitic secondary infections. Chronic diarrhoea from chronic gastrointestinal infections leads to chronic iron deficiency anaemia and to the distribution and frequency of porotic hyperostosis visible in prehistoric Anasazi skeletal populations.

The preceding sections indicate the role of chronic bacterial, viral and parasitic infections in creating and perpetuating iron deficiency anaemia,⁴ especially in children under the age of five. A horticulturalist's diet need not be the primary factor in explaining the geographical distribution, frequency and age distribution of porotic hyperostosis and iron deficiency anaemia, since most prehistoric Southwestern people ate maize *in combination with* beans, squash, plants now wild, wild plants and meat. This is not to deny any relationship between a solely vegetarian, and especially maize, diet and anaemia in those areas where such a diet existed. One such area may have been the Valley of Mexico during the Classic and post-Classic periods of high population densities (Santley, personal communication). I believe, however, that the prehistoric Southwest was *not* one of those areas.

The link between diarrhoeal diseases and anaemia is through the nonabsorption of all the available nutrients ingested and/or blood loss, usually in the stools. The causes of diarrhoeal diseases are those that are accentuated and perpetuated in sedentary aggregated settlements (Schliessmann 1959: 381). Although not always recognised in the prehistoric Southwest, the effects of sedentism and aggregation have been postulated as the impetus behind the frequency of prehistoric diseases in the rest of the world (e.g., Polunin 1967: 74–75). For instance, beginning with the earliest villages:

Certain contagious diseases of man maintain their reservoir of infection solely by a continuous chain of contact between infective and susceptible persons. . . . Many of these viruses and pathogens have short persistence outside the human body and it is, therefore, evident that a successful chain of infection must depend to a large degree on how close the susceptible persons are to the infected ones (Ipsen 1959: 162; see also Cockburn 1959; 1971).

The aggregation and sedentism characteristic of the Mesolithic and Neolithic in various parts of the world provided new breeding places for many forms of life which harbour disease (Armelagos & Dervy 1970: 273; also see Polgar 1964). Although perhaps less a factor among the Anasazi, the general incidence of parasites also increases among sedentary aggregated people. A study of nomadic Basarwa (Bushmen or San) shows that, with the exception of hookworm (*Necator americanus*), they are relatively free of parasites compared to their more sedentary and aggregated Bantu-speaking neighbours who are plagued by a number of different parasites including pinworm (*Enterobius vermicularis*), small tapeworms (*Strongyloides stercoralis*), Hydatid cyst (*Echinococcus granulosum*), large roundworm (*Ascaris lumbricoides*), and whipworm (*Trichocephalus trichura*) (Heinz 1961: 209–11). It is the Basarwa's nomadism and small group sizes that help deter parasitic infections. Studies also show that the age range of the highest

incidence of porotic hyperostosis in archaeological skeletal populations is isomorphic with the age range of the most susceptible parasite hosts, that is infants and children under ten years of age: 'It is well known that juveniles are above all endangered by worm infestations. Thus for instance in several North African areas practically 100% of the children suffer from helminthiasis' (Hengen 1971: 68).

Perhaps most interesting and compatible with the proposed nondiet cause of porotic hyperostosis in the prehistoric Southwest is the fact that despite their high meat diet, Eskimo skeletal populations have high percentages of porotic hyperostosis that are sometimes even higher than for some Southwestern horticultural groups. In one study, 20.2 per cent. of ninety Eskimo crania exhibited evidence of porotic hyperostosis (Nathan 1966). Obviously a meat or iron deficient diet cannot account for this relatively high incidence of porotic hyperostosis among Eskimos, since they are known for their reliance on a meat-dominated diet. The ramifications of sedentism and aggregation can, however, account for this high percentage, since Eskimos traditionally occupied seasonally sedentary winter villages. Parasitic infections are known to afflict Eskimos, some of which could potentially cause iron deficiency anaemia (e.g., Schiller 1951; see also Rausch 1951: 114 for a discussion of the presence of *Echinococcus granulosus*, a small tapeworm). Bacillary infections producing diarrhoea also are endemic to Eskimos. Such infections as *Shigella flexneri* and *Salmonella typhosa* which often cause diarrhoea are common in historic Eskimo villages, some of which have as many as one-third to one-half of the occupants ill with diarrhoea at any one time during the year, with children the most frequent victims (Oswalt 1967: 76-7). Furthermore, prehistoric marine-dependent Californian Indians had a similar frequency of porotic hyperostosis to horticulturalists despite the former's heavy reliance on fish (Walker 1986).

It is informative to compare in detail the semi-sedentary or seasonally sedentary Eskimo hunters and gatherers with the nomadic !Kung Basarwa hunter-gatherers. The !Kung 'have an unusual system of sanitation. They live about a mile from their water and defecate into the sand some distance from their camps' (Silberbauer 1965). The feces, rapidly dried by the sun, are disposed of by dung beetles. Furthermore, the San are not crowded and move camp several times a year' (Truswell & Hansen 1976: 171). Significant, and I believe consequent, is the fact that the !Kung are rarely anaemic, despite the occasional hookworm infection evident in three of eighteen faecal samples (Truswell & Hansen 1976). Anaemia was 'present in only one sickly infant with malaria (anaemia caused by a chronic disease), one man out of 38 and one woman out of 113 nonpregnant females' (Truswell Hansen 1976: 186). It is important to note that the !Kung children 'are not anemic even though they are breast fed until they are three years old or older' (Truswell & Hansen 1976: 193-194); prolonged lactation often being blamed for causing porotic hyperostosis in prehistoric populations (also questionable is how exclusively breast milk actually was in an Anasazi youngster's diet after the first year). Pygmies, another nomadic hunting and gathering group, also show no signs of iron deficiency anaemia (Truswell & Hansen 1976: 192).

Although a higher mortality rate from a variety of causes was recorded for the

nomadic versus the newly sedentary !Kung (Harpending & Wandsmider 1982), sedentary !Kung tend to appear outwardly less healthy than the still nomadic ones (Harpending, personal communication). In fact, Hitchcock has noted that:

Finally, some observations on the nutritional status of mobile and sedentary Basarwa populations in eastern Botswana reveal that nutritional problems are greater in settled situations. . . . Poor hygienic conditions and nutritional problems often correlate with high rates of infective diseases (Robson and Wadsworth 1977: 196). Outbreaks of epidemic disease occurred in the Kalahari in 1950-1 and as recently as July 1977. . . . In spite of the trend toward greater stature, both the health and the nutritional statuses of settled Basarwa populations were poorer than those of mobile ones. . . . (1982: 254-5)

Australian Aborigines in settled communities had iron deficiency anaemia 10 to 20 times more often than non-Aborigine children in Australia (Taylor 1977: 147).

The malnourishment of infants and children [in the settled communities] was not simply a matter of their inadequate food intake. Intestinal malabsorption resulting from frequent bouts of infection . . . was also implicated. . . . [At the settlement, children] acquired repeated respiratory and gastrointestinal infections which, if they did not prove to be fatal, may have impaired the children's ability to absorb nutrients from the diet (Taylor 1977: 147-8).

The presence of Harris lines, transverse trabeculae or radio-opaque lines (Platt & Stewart 1962), is often attributed to dietary stress and used to further the dietary model for the changes in porotic hyperostosis frequencies (see Huss-Ashmore *et al.* 1982: 434-41). They can also occur, however, after the stress of a severe illness not related to a person's diet. After such an illness, *regardless of the normal diet*, Harris lines can develop during childhood because the recuperating body requires an increase in the amount of iron consumed, which usually occurs at a time when a person actually desires less nourishment because of nausea. Other studies also indicate that Harris lines do not develop only as the result of chronic dietary deprivation (e.g., Buikstra 1976: 356). In fact, Harris lines may be the result of slight subclinical infections and children 'with no apparent history of severe illness may possess similar lineations . . . further indicating that a trivial condition may motivate the production of a Harris line' (Lobell 1984: 110). The presence of Harris lines can vary between observers, radiographic techniques used, skeletal element observed, and age of sample (Goodman *et al.* 1984: 24). In addition, new data reveal that there are 'low correlations between Harris lines and known stressors in the clinical and experimental literature as well as a series of inverse relationships between Harris lines and other stress indicators in paleopathological studies' (Goodman *et al.* 1984: 24). Also, any stress from diarrhoeal infectious diseases can result in stress of great enough magnitude potentially to create Harris lines. Clearly more research is needed.

Although combinations of disease factors have been suggested as causing the abandonment of specific sites and of parts of the Southwest in general (e.g., Colton 1936; Kunitz & Euler 1972; Palkovich 1984; Ryan 1977), that is too simplistic a model for a very complex phenomenon. I would concur that the increase in chronic diseases may have had some effect, but *only in combination* with social, political, economic, and in some areas, environmental changes and

stresses during the later periods, which together resulted in the abandonment of large parts of the Southwest region. That does not negate the influence of disease in the prehistoric Anasazi and other groups, but it does recognise the limitations of it as a prime mover explanation. Certainly Europe, with its present-day high incidence of diseases (specifically anaemia) was not abandoned as a result of massive epidemics. While it is necessary to recognise the importance of disease, it is equally important not to over-emphasise its role in the culture history of any one area.

Conclusions and summary

The incidence of porotic hyperostosis in North American southwestern Anasazi populations has in the past been attributed to a maize-dependent diet, which is one low in iron, although a few anthropologists have suggested that it was in combination with infectious diseases. This article has shown: (1) that the prehistoric Anasazi diet was not primarily dependent upon plant species—particularly maize—and that this is probably true for many groups around the world prior to European/Euroamerican intervention; (2) that the Anasazi diet was probably nutritionally adequate and contained sufficient iron for normal and healthy people, the incidences of porotic hyperostosis being the result of different factors; (3) that Anasazi children were anaemic as a result of the diseases that go along with sedentism and aggregation, the percentages not differing that much from the percentages of anaemic children present in modern Third World countries; and (4) that these diseases include chronic bacterial, viral and parasitic infections which cause chronic diarrhoea and/or blood loss and lead to chronic iron deficiency anaemia.

It is hoped that this article will raise serious questions about the common assertion that the presence of porotic hyperostosis in southwestern Anasazi skeletal populations is the result of a nutritionally poor diet, possibly aggravated by unspecified infectious diseases. The ramifications of sedentism and aggregation primarily in terms of endemic chronic infections best explain the distribution of anaemia and porotic hyperostosis in a number of regions. When contrasted with modern and other prehistoric populations, the incidence of iron deficiency anaemia among the Anasazi is not as high or unusual as one might first suspect. Such anaemia is present, sometimes at high levels, in most Western European countries, as well as in developing nations.

To assume *a priori* and uncritically that diet was always a causal factor to one degree or another in iron deficiency anaemia is as careless thinking as assuming without evidence that diet was never a factor. It is now incumbent upon the diet model champions systematically to demonstrate that their position better accounts for the observed patterns of porotic hyperostosis and better explains the data than the sedentism-aggregation model in any particular area. Finally, if we are to see the sedentism and aggregation of people in the form of villages and towns as a growth in the complexity of human organisation, we must also view it as a growth in human pestilence.

We can now begin to formulate predictive models for newly sedentary groups

such as the !Kung Basarwa and their future, as well as to develop models that allow us to understand the past more realistically and the processes that influenced it. Implications of this model reach far beyond the health status of an ancient population to question current priorities in some Third World countries' development funding which is oriented towards changing diet as part of some international health projects. It will be most interesting to note the future effects of sedentism and aggregation on the newly sedentary Basarwa and Aborigines after a few generations have passed. We should be able to learn from the past, as well as the present, and modify funding programmes to emphasise the eradication of infectious diseases rather than concentrate so much on altering food consumption and agricultural practices.

NOTES

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¹ It is sometimes forgotten that humans do not always make decisions based on calculations of the amount of caloric energy expended compared to that obtained from an activity. The effort placed on the widespread ceramic exchange system operating throughout the Anasazi Southwest illustrates this, since with few exceptions, the inhabitants could have expended energy more efficiently through the use of only locally produced plain pottery. Instead, they also made corrugated and painted wares and sought others from far and wide. Surely people today do not have to be told that humans are not always rational in their actions nor do they always maximise the most for the least effort, and trade and/or long treks to obtain meat and other items was probably more common prehistorically than is currently acknowledged.

² I have admittedly not dealt with the issue of dentition for reasons of space, even though Anasazi skeletons contain various sorts of dental pathologies. Some of these were affected by the roughage of their diet, others by heredity (e.g., the hardness of the enamel), the use of their teeth for tasks not connected with eating (e.g., chewing hides), the amount of intrusive nonedible particles unintentionally added to their food (e.g., sand and rock from grinding food on metates), the amount of fluorine and other minerals in their drinking water, and the general health of the people (e.g., sickly individuals with infections and anaemia would probably be more susceptible to dental disease than healthy persons).

³ Diet can play a role in permitting the infestation of certain protozoa, such as amoebas, that in high meat diets are unfavourable (Chandler & Reed 1961: 72). Although speculative, nomadic Palaeo-Indians may not have suffered as much from amoebas or other such infections, whereas sedentary and aggregated populations, anaemic despite their diet due to bacterial, viral and parasitic infections, may have been more susceptible to infestation because the iron ingested was not absorbed. This would have led to further malnutrition, regardless of diet and could have eventually led to premature death.

⁴ According to Lee *et al.* (1980: 1515), iron deficiency anaemia can result from a variety of secondary causes, including most importantly menorrhagia and secondarily ulcers, neoplasm, haemorrhoids, etc. (also see Madden & Wilson 1980: 224–5; Rybo 1970: 169–70). Fevers from infections can potentially accelerate the loss of iron through excessive sweating (Bothwell 1970: 156) and be a contributing factor. Pregnancy also makes demands on iron stores, especially during the latter half of term (Miale 1982: 412; Duffy 1980: 15), although these tend to be either more minor or idiosyncratic causes of iron deficiency anaemia.

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